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SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R) 1950-2006/Dec 16
        (c) format only 2006 Dialog
*File 155: MEDLINE has resumed updating with UD20061209. Please
see HELP NEWS 154 for details.
  File  55:Biosis Previews(R) 1993-2007/Jan W3
        (c) 2007 The Thomson Corporation
  File  34:SciSearch(R) Cited Ref Sci 1990-2007/Jan W3
        (c) 2007 The Thomson Corp
  File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
        (c) 2006 The Thomson Corp
  File 340:CLAIMS(R)/US Patent 1950-07/Jan 25
        (c) 2007 IFI/CLAIMS(R)
*File 340: The 2006 reload is online as of December 1, 2006.
IPCR/8 is available.

      Set  Items  Description
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? s prb(2n)bind?
          10062  PRB
        2392327  BIND?
      S1    921  PRB(2N)BIND?
? s casein(w)kinase(w)ii(5n)phosphoryl?
          70955  CASEIN
         866055  KINASE
        2053110  II
         504936  PHOSPHORYL?
      S2   2739  CASEIN(W)KINASE(W)II(5N)PHOSPHORYL?
? s s1 and s2
            921  S1
           2739  S2
      S3     26  S1 AND S2
? s (second or two) (5n) casein
Processing
        3532539  SECOND
        7531858  TWO
          70955  CASEIN
      S4   1476  (SECOND OR TWO) (5N) CASEIN
? s s3 and s4
             26  S3
           1476  S4
      S5      1  S3 AND S4
? t s5/3,k,ab/1

 5/3,K,AB/1     (Item 1 from file: 340)
DIALOG(R)File 340:CLAIMS(R)/US Patent
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Dialog Acc No: 10632258
IFI Chemical Acc No: 2004-0040710
Document Type: C
CDNA ENCODING A GENE BOG (B5T OVER-EXPRESSED GENE) AND ITS PROTEIN PRODUCT;
TUMOR SUPPRESSION LIGAND COMPRISING RETINOBLASTOMA AND CASEIN BINDING
MOTIVES FOR USE TREATMENT OF CANCER; ANTITUMOR AGENTS
Inventors: Thorgeirsson Snorri S (US); Woitach Joseph T (US); Zhang
    Minghuang (US)
Assignee: U S of America Health & Human Services
Assignee Code: 06814
Attorney, Agent or Firm: Attention: Katherine M. Kowalchyk;MERCHANT & GOULD
    P.C., P.O. Box 2903, Minneapolis, MN, 55402-0903, US
Publication (No,Kind,Date), Applic (No,Date):
US 20040139485 A1 20040715 US 2004772988  20040205
Continuation Pub(No),Applic(No,Date): Pending                    WO
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99US4142 19990225
Division Pub(No),Applic(No,Date): US 6727079 US 2000637746 20000811
Priority Applic(No,Date): US 2004772988 20040205; WO 99US4142 19990225; US 2000637746 20000811
Provisional Applic(No,Date): US 60-75922 19980225; US 60-79567 19980327

The human papillomavirus E7 oncoprotein and the cellular transcription factor E2F bind to separate sites on the retinoblastoma tumor suppressor protein.

Wu E W; Clemens K E; Heck D V; Munger K
Laboratory of Tumor Virus Biology, National Cancer Institute, Bethesda, Maryland 20892.

Journal of virology (UNITED STATES) Apr 1993, 67 (4) p2402-7, ISSN 0022-538X--Print Journal Code: 0113724
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

The ability of the high-risk and low-risk human papillomavirus E7 oncoproteins to disrupt complexes of the retinoblastoma tumor suppressor protein pRB and the cellular transcription factor E2F was studied. The ability of E7 to disrupt this transcription factor complex correlated with the different pRB binding efficiencies of the high-risk and low-risk human papillomavirus-encoded E7 proteins. The ***pRB*** ***binding*** site was the sole determinant for these observed differences. The phosphorylation status of the casein kinase II site that is immediately adjacent to the pRB binding site in E7 had no marked effect on this biochemical property of E7. Peptides consisting of the pRB binding site of E7, however, were not able to disrupt the pRB/E2F complex. These data suggest that additional carboxy-terminal sequences in E7 are also required for the efficient disruption of the pRB/E2F complex and that E7 and E2F may interact with nonidentical sites of pRB.

Analysis of the interaction between human papillomavirus type 16 E7 and the TATA-binding protein, TBP.

Phillips A C; Vousden K H
ABL Basic Research Program, NCI-FCRDC, Frederick, MD 21701-1201, USA.

Journal of general virology (ENGLAND) Apr 1997, 78 (Pt 4) p905-9,
ISSN 0022-1317--Print Journal Code: 0077340
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

The E7 protein encoded by human papillomavirus type 16 shows transforming and immortalizing activities which are mediated, in part, through the interaction of the viral oncoprotein with the pRB protein family. This interaction is not solely responsible for E7 function, however, and other properties of E7, such as the interaction with basal transcription factors such as TBP, are likely to be of importance. We show here that three regions of the viral protein contribute to the interaction between E7 and TBP; the pRB-binding domain, the casein kinase
II phosphorylation region and the C-terminal dimerization
domain. Mutations within each region reduced the interaction of E7 with TBP in vitro, and simultaneous alterations within each of these regions completely abrogated binding. Unlike the pRB interaction, the association of E7 with TBP was enhanced following phosphorylation of E7 by
casein kinase II, demonstrating a functional significance
for ***phosphorylation*** of the viral protein.

4/3,K,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11580832 PMID: 9426450

Destabilization of the RB tumor suppressor protein and stabilization of p53 contribute to HPV type 16 E7-induced apoptosis.

Jones D L; Thompson D A; Munger K

Program in Biological and Biomedical Sciences, Harvard Medical School, Boston, Massachusetts 02115, USA.

Virology (UNITED STATES) Dec 8 1997, 239 (1) p97-107, ISSN 0042-6822--Print Journal Code: 0110674

Contract/Grant No.: CA 66980; CA; NCI; ES 07155; ES; NIEHS

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Cells that express the human papillomavirus (HPV) type 16 E7 oncoprotein are predisposed to undergo apoptosis. Transgenic mice that have E7 expression targeted to either the retinal photoreceptor cells or the lens cells exhibit signs of apoptosis in cells attempting to undergo differentiation. We established a cell culture system to study this process and have determined the domains of E7 that are required for predisposing cells to undergo apoptosis in response to growth arrest signals. Regions within the core pRB binding site of E7 were necessary but not sufficient for inducing apoptosis. Residues within the adenovirus conserved region 1 homology domain and the consensus casein kinase II phosphorylation site are also important for this effect on cell viability. Our data also demonstrate that the ability of E7 to induce destabilization of pRB and stabilization of p53 coincides with E7-mediated transformation and apoptosis.

... predisposing cells to undergo apoptosis in response to growth arrest signals. Regions within the core ***pRB*** ***binding*** site of E7 were necessary but not sufficient for inducing apoptosis. Residues within the

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*File 340: The 2006 reload is online as of December 1, 2006.
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      Set  Items  Description
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? s prb(w)binding
           10062  PRB
         2166351  BINDING
      S1     424  PRB(W)BINDING
? s casein(w)kinase(w)II(5n)phosphorylat?
           70955  CASEIN
          866055  KINASE
         2053110  II
          461298  PHOSPHORYLAT?
      S2    2725  CASEIN(W)KINASE(W)II(5N)PHOSPHORYLAT?
? s s1 and s2
             424  S1
            2725  S2
      S3      19  S1 AND S2
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